EFFECTS OF AQUATIC HERBICIDES ON FISHERIES

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Summary Fish populations are at risk from the use of aquatic herbicides. Acute toxicity data alone are insufficient to assess these risks and the minimum data required have been given in the paper. These data form a part of the Pesticides Safety Precautions Scheme requirements for aquatic clearance. Data on all the aquatic herbicides cleared for use in the U.K. have been presented and an assessment of the risk to fisheries has been made.

INTRODUCTION

Aquatic weed clearance is carried out annually along most of the water-courses managed by the Internal Drainage Boards and 30% of the main river systems (Robinson, 1971), because of the need to prevent weed growth which impedes water flow and causes problems in flood prevention, navigation and water supply. Large areas of static water are also subjected to annual weed clearance, because of the increased need for open-water by a growing number of water-based recreational activities. The use of chemicals to control aquatic weeds in these waters has increased over recent years because of the development of better formulations and more favourable costs compared with mechanical and manual methods. Although the use of herbicides may not greatly reduce the overall costs of weed removal, it does allow a larger area to be free of weeds earlier in the season than would otherwise be the case, which may be important from a flood prevention point of view (Robinson, 1971). The first chemicals to be used for aquatic weed control were existing formulations of terrestrial herbicides but over recent years granular formulations have been developed to produce a slower release of chemicals, thereby increasing their persistence in water as well as allowing easier and more accurate application. Therefore, it has become possible, and in certain areas desirable, to apply such chemicals to large areas during a single growing season. Naturally occurring fish populations as well as those in managed fisheries are potentially at risk from the chemicals used for aquatic weed clearance.

When a herbicide is applied to a water body the active ingredient is liberated for a sufficient time to cause the eventual death of the target weed. Herbicide residues can be lost from water by volatilisation, photo-decomposition, degradation or adsorption onto plant or mud material. After

herbicide application fish are exposed to an initial increase and then a decline in aqueous residue concentration, possibly followed by a further increase as the plants decay and release residues. These residues will be taken up by the fish during the exposure period and possibly accumulated in the tissues. The degree of accumulation, and therefore their physiological effect, will depend on the rate of elimination of the residues, either as the original molecule or as a metabolite. As the level of herbicide to which the fish are exposed falls, so the residue levels in their tissues will decrease at a rate depending on the ability of fish to eliminate the residues.

Some measure of the risk to fish from the use of a herbicide may be obtained from the rate at which the residues are taken up and accumulated in the tissues and the rate at which they are eliminated when fish are subsequently placed into clean water. If the herbicide residues are persistent in fish tissue then sub-lethal or chronic effects may occur. Persistent residues in mud may be accumulated by benthic organisms and in turn by those organisms which feed upon them; however, effects on the food web are difficult to assess. After normal treatment of a water body with an aquatic herbicide, fish will accumulate residues from the water by uptake across the gills and it is thought that dietary intake plays only a small part in the total residue accumulation.

At low exposure concentrations the normal fish detoxification and elimination mechanisms will cope with residue uptake, and homeostasis will be maintained. Increased residue uptake may impair some biochemical or physiological function and if exposure is maintained for a long enough period, or uptake is further increased, then the fish may not be able to compensate for the impairment and permanent disability or death may occur. This series of events can be applied to the cellular level as well as to the community level of biological organisation.

The Pesticides Safety Precautions Scheme (P.S.P.S.) (MAFF 1966), further assesses the operator, consumer and environmental safety of biocides, and gives clearance for use if they pass the necessary standard. The type of fish toxicity data required from the pesticide manufacturer depends on the use and extent of application of the chemical. Aquatic herbicides, as they are used in or near water, require more extensive fish toxicity and residue data than for many other pesticides. Although data from acute fish toxicity tests alone may be sufficient for terrestrial herbicide clearance, they are inadequate for aquatic clearance. The overall requirements for registration of aquatic herbicides have been discussed in a previous paper (Bates, 1976). To assess the possible risks to fish the minimum data required are as follows, and they have been included in the new Working Document No. 6 of the P.S.P.S.

(a) Acute toxicity using an appropriate test species.

(b) Rates of accumulation and elimination of herbicide residues in tissues of rainbow trout (*Salmo gairdneri*) exposed to the normal application concentration, or concentrations corresponding to 10% and 50% of the 96 hour LC50, for a period and subsequently placed in clean water.

(c) Residue analyses of any fish that died after exposure to the chemical in order to determine the tissue concentration associated with death.

If the margin of safety is small or the chemical is persistent in the fish tissues or in the environment additional data may be required from field trials. Such data would establish the tissue residue levels in fish, water and mud after exposure to the herbicide when applied at the recommended application dose, under the fluctuating temperature and dissolved oxygen regimes encountered in practice. Further experiments may be required using a different fish species or a longer period of exposure to establish the chronic toxicity.

In this paper, an assessment will be made of the risk to fisheries from the herbicides used or intended to be used in this country for weed control in or near water, using data obtained by the Salmon and Freshwater Fisheries Laboratory and from other published evidence.

The herbicides used for weed clearance in or near water and the type of weeds they control are given in Table 1. Included in this Table are the maximum recommended application rates for herbicides used to control submerged weeds and, for herbicides used to control emergent and bankside weeds, the maximum concentration which would be found after applying the herbicide at the recommended rate direct to water 1 metre deep.

TABLE 1 Aquatic herbicides used in U.K.

Chemical	Weeds controlled	Recommended application concentration (mg/l)	Maximum concentration if applied to water 1 metre deep (mg/l)
Asulam	Emergent		1.0
Chlorthiamid	Submerged	1.0	
Copper Sulphate	Algae	1.0	
Cyanatryn	Submerged and Algae	0.2	
2,4-D (amine)	Bank Weeds		0.4
Dalapon	Emergent		3.0
Dichlobenil	Submerged	1.0	
Diquat	Submerged	2.0	
Glyphosate	Emergent		0.6
Maleic Hydrazide	Bank grass		1.5
Paraquat	Emergent and submerged	1.0	
Terbutryne	Submerged and algae	0.1	

Of these herbicides Prefix (chlorthiamid), 2,4-D (amine), Dalapon (aquatic formulation), Casoron (dichlobenil), Aquacide (diquat), Maleic Hydrazide, Esgram (paraquat) and Clarosan (terbutryne) have been cleared for aquatic use by the P.S.P.S., and Asulox (asulam), Aqualin (cyanatryn) and Roundup (glyphosate) are under consideration. Copper sulphate has not been cleared but because of its algicidal properties it is still used in this country.

TOXICITY OF AQUATIC HERBICIDES TO FISH AND INVERTEBRATES

Acute Toxicity

The acute toxicity of herbicides to a wide range of fish and invertebrate species is well documented (Hughes and Davis, 1962, 1963, 1964; McKee and Wolf, 1963; Cope, 1965, 1966; Sanders and Cope, 1966, 1968; Alabaster, 1969; Wilson and Bond, 1969; Mullison, 1970; Sanders, 1970; Kemp *et al*, 1973; Tooby, 1971; Walker, 1971; Tooby, *et al*, 1974, 1975; Tyson, 1974). It is generally accepted that, in order to obtain reproducible results from fish toxicity tests, constant flow test conditions should be used. However, few of the results reported were from such tests, the majority being obtained under static conditions. In the U.K. a standard constant flow fish toxicity test procedure has been used for many years, with the harlequin fish (*Rasbora heteromorpha*) as the test species (Alabaster and Abram, 1965). This fish was chosen because it has a similar sensitivity to the rainbow trout (*Salmo gairdneri*) for a wide range of poisons. Results from the U.K. test using the harlequin fish in soft water are not at variance with the published acute toxicity data for other species of fish. For simplicity only the results of the U.K. test are given in Table 2. All the herbicides were tested under the same conditions and therefore a comparison can be made of their toxicity. The commercial formulation of each herbicide was tested to take into account any effect on the toxicity of the active ingredient by other formulated chemicals and the results expressed as the concentration of total formulation, except for chlorthiamid, cyanatryn, dichlobenil and terbutryne, where the technical grade was used instead of the granular formulation. The median lethal concentrations (LC50) for 24, 48 and 96 hours have been calculated together with the 10 percentile lethal concentrations using the methods of Litchfield and Wilcoxon (1949). A margin of safety has been calculated from the difference between the 96-hour LC10 and the maximum concentration likely to be found after application of the herbicide to water at the recommended rate.

The acute toxicity of the herbicides to some invertebrates is given in Table 3, including data obtained by the Salmon and Freshwater Fisheries Laboratory. Not all the tests were conducted with the same formulations as those used for the fish toxicity tests. For example, the results for 2,4-D are from a test using an ester formulation which is much more toxic than the U.K. recommended amine formulation. Some species of invertebrates seem to be particularly sensitive to herbicides at concentrations below those found in water after recommended treatment. The margin of safety for copper sulphate is nil and very small for dichlobenil (and presumably for chlorthiamid as this chemical quickly breaks down to dichlobenil in water). Overall it seems that the margin of safety for invertebrates is less than that for fish.

TABLE 2 Acute toxicity of aquatic herbicides to fish and their margin of safety

Trade name	Active ingredient	% composition	Lethal concentrations (mg/l)							Margin of safety
			24 hour		48 hour		96 hour		Extrapolated	
			LC10	LC50	LC10	LC50	LC10	LC50	3 month LC50	
ASULOX	ASULAM	40	-	>5000	-	>5000	-	>5000	-	>5000
PREFIX	CHLORTHIAMID	91	33	41	30	34	27	32	20	27
	COPPER SULPHATE	100	-	<1.0	-	<1.0	-	<1.0	-	0
AQUALIN	CYANATRYN	100	15	35.5	9	18.5	9.5	15	5	48
	2,4-D(amine)	50	1825	1900	1320	1425	1150	1250	800	2875
	DALAPON	85	6600	8900	4400	6400	3800	5200	4000	1267
CASORON	DICHLOBENIL	100	13	16	9	12	5	6.5	1.0	5
AQUACIDE	DIQUAT	13.5	370	450	265	350	162	252	100	81
ROUNDUP	GLYPHOSATE	36	17	21	11	16	9	12	8	15
	MALEIC HYDRAZIDE	36	300	530	-	150	-	125	100	> 50
ESGRAM	PARAQUAT	20	30	47	20.5	35	15	25	10	15
CLAROSAN	TERBUTRYNE	100	12	15	10.5	14	7	11	5	70

TABLE 3 The acute toxicity of aquatic herbicides to aquatic invertebrates

Herbicide	Species	LC50 (mg/l)			Reference
		24 hr	48 hr	96 hr	
Asulam	*Gammarus spp.*	-	-	15000	
	Asellus spp.	-	-	30000	
Copper Sulphate	*Ephemeralla spp.*	-	0.32	-	Kemp *et al* (1973)
	Daphnia magna	-	-	0.096	McKee and Wolf (1963)
2,4-D	*Pteronarcys californica*	-	1.8	1.6	Sanders and Cope (1968)
	D. pulex	-	3.2	-	Walker (1971)
	Simocephalus serrulatus	-	4.9	-	Walker (1971)
Dalapon	*P. californica*	-	-	100	Sanders and Cope (1968)
	D. pulex	-	11	-	Sanders and Cope (1966)
	S. serrulatus	-	16	-	Sanders and Cope (1966)
Dichlobenil	*Hyalella azeteca*	-	12.5	8.5	Wilson and Bond (1969)
	Callibaetis spp.	19.2	10.3	-	Wilson and Bond (1969)
	Limnephilus spp.	28.2	23.3	13.0	Wilson and Bond (1969)
	Enallagma spp.	25.5	24.2	21	Wilson and Bond (1969)
	Tendepedid midge larvae	15.7	-	7.8	Wilson and Bond (1969)
	P. californica	42	8.4	6.6	Cope (1966)
	S. serrulatus	-	5.8	-	Cope (1966)
	D. pulex	-	3.7	-	Cope (1966)
	D. magna	9.2	7.8	-	
	Gammarus pulex	5.4	3.2	2.4	
	Asellus aquaticus	7.2	4.7	2.9	
Diquat	*H. azeteca*	-	0.12	0.048	Wilson and Bond (1969)
	Callibaetis spp.	-	65	33	Wilson and Bond (1969)
	Limnephilus spp.	-	100	100	Wilson and Bond (1969)
	Enallagma spp.	-	100	100	Wilson and Bond (1969)
Paraquat	*S. serrulatus*	-	4.0	-	Sanders and Cope (1966)
	D. pulex	-	3.7	-	Sanders and Cope (1966)
Terbutryne	*Chironomus thummi*	-	5-10	-	Tyson (1974)
	Aedes aegypti	-	10	-	Tyson (1974)
	D. magna	-	1.4	-	Tyson (1974)
	Tubifex tubifex	-	10	-	Tyson (1974)

Chronic Toxicity

Data on chronic toxicity and analysis of the tissue residue level associated with death have not been as well documented as that for acute toxicity. The median survival times for rainbow trout and roach (*Rutilus rutilus*) exposed to 1.0 mg/l of dichlobenil (recommended application concentration) were 58 ± 5 days and 26 ± 4 days respectively (Tooby, *et al*, 1974). During this study it has shown that the test fish refused food and swam slowly around the aquaria whereas the control fish accepted food and swam violently away from any shadows cast over the aquaria. The test fish lost condition and their fat reserves were reduced. In similar studies, guppies (*Lebistes reticulatus*) swam slowly around the aquaria during exposure to 10 mg/l of dichlobenil (Niehuss and Börner, 1971a), and goldfish (*Carassius auratus*) swam with jerking movements and irregular respiration in 6.4 mg/l (Bolier *et al*, 1974). The average residues in trout tissue at death after the chronic exposure were 2.4 ± 0.6 mg/kg in muscle, 7.9 ± 0.9 mg/kg in liver and 12.3 ± 6.5 mg/kg in brain and, for roach muscle, liver and brain, 4.0 ± 2.9 mg/kg, 12.3 ± 1.7 mg/kg and 20.7 ± 3.3 mg/kg respectively. These lethal tissue residue concentrations after the chronic exposure were about 10 times lower than the acute lethal tissue concentration in trout and about three times lower than that for roach and may be associated with the long-term reduction in fat reserves.

Toxicity of herbicides to fish eggs and fry

It is important to assess the risks from the use of herbicides to the juvenile stages of fish as well as the adults. Apart from the effects of weed removal on feeding and predation, there is the probability that eggs or newly hatched fry may be more susceptible to herbicide residues than the adults. Hiltibran (1967) showed that 25 mg/l 2,4-D (amine) had no effect on the eggs or fry of bluegills (*Leopomis machrochirus*). Similarly, dalapon concentrations of up to 50 mg/l and dichlobenil up to 10 mg/l had no effect on bluegill fry survival, but the fry survived for only three days in 2.4 mg/l of diquat. However, these tests were made under static conditions and in the case of dichlobenil a granular formulation was tested so that the concentration would have taken some time to reach the maximum level.

ACCUMULATION AND PERSISTENCE OF HERBICIDE RESIDUES IN FISH, MUD AND WATER

Fish exposed to herbicides in water will take up residues, which may accumulate in the tissues. It is necessary, therefore, to measure the rate of uptake and elimination of the herbicide residues in order to obtain information on the persistence within the tissues and also whether a lethal tissue residue level could be reached after normal herbicide application. Residues may quickly reach a plateau level, which may be lethal if present for a sufficient time, or may gradually increase until a tissue level is reached which is then rapidly lethal. Therefore, studies on uptake rates should be ideally carried out in constant exposure conditions, followed by a period in clean water, with

fish samples taken at intervals during the exposure and clean water period for residue analysis. However, few experiments have been conducted from which rates of accumulation and elimination can be obtained. Most of the published work refers to results from field studies.

It is important to determine herbicide levels in fish, water and mud after a water body, or the vegetation around it, has been treated at the recommended application rate, in order to establish the effects of the herbicide under normal field conditions. Usually the concentration found in water after the recommended dose has been applied, is lower than the nominal concentration. One effect of herbicide application to control submerged plants is to reduce the dissolved oxygen concentration by inhibition of photosynthesis and decomposition of the plants. The decomposition of a large plant biomass would greatly reduce the dissolved oxygen concentration to a level which may be lethal to fish. However, the dissolved oxygen concentration normally found after herbicide treatment is usually not lethal for those coarse fish species found in areas likely to receive herbicide treatment, but it could enhance the effect of the herbicide on fish. Treatment, of emergent weeds is less likely to cause this effect unless the herbicide is applied directly on to water. There may also be a decrease in pH of the water associated with increased bicarbonate alkalinity and free carbon dioxide (Brooker and Edwards, 1975).

Toxicologically, long exposures to low herbicide concentrations may be as disadvantageous to fish as high concentrations for short exposure periods. With the trend towards granular formulations of aquatic herbicides for submerged weed control the persistence of herbicide residues has been increased due to the slow-release properties of this type of formulation.

Data on aquatic herbicides

The following data on the accumulation and persistence of herbicide residues in fish, water and mud are presented under separate headings for each herbicide.

ASULAM

This chemical is unlikely to be applied directly onto water so the residues will not reach the maximum concentration given in Table 1; furthermore, the margin of safety for aquatic life is large. Ingham and Gallo (1975) reported that because of its high water solubility little or no accumulation of asulam residues would take place.

COPPER SULPHATE

From laboratory experiments, Brungs *et al* (1973) showed that after exposure to concentrations up to 0.1 mg/l copper was accumulated slowly up to 30 days, after which equilibrium was reached in gill, liver and kidney tissues of brown bullheads (*Ictalurus nebulosus*). No significant difference was found

between the copper levels in live fish after sub-lethal exposure and levels in fish which died from copper poisoning. Copper persistence in water depends on the water hardness, pH and organic content. Although the margin of safety calculated from laboratory tests is nil, in practice copper is precipitated or complexed to organic molecules or is accumulated in the mud by adsorption, thereby increasing the margin of safety. In one field study a pond was treated with 3 mg/l of copper in the form of copper sulphate; the aqueous concentration declined rapidly and copper accumulated in the mud and remained there for the duration of the four-month study (McIntosh, 1975). After one week green-sunfish (*Lepomis cyanella*) had accumulated an equilibrium concentration of 3 mg/kg of copper in their tissues. This plateau concentration, which was lower than the tissue residue levels reported from fish that had died from copper poisoning (Brungs *et al*, 1973), remained for 47 days after treatment, and decreased to the background level by 79 days. Copper remained in the fish for five weeks longer than in the pond water.

CHLORTHIAMID AND DICHLOBENIL

Chlorthiamid breaks down quickly to dichlobenil in water, although it is more stable in acid conditions (Beynon and Wright, 1972). Both chlorthiamid and dichlobenil are adsorbed onto mud where they remain biologically active. In laboratory experiments it has been shown that dichlobenil is accumulated in fish tissues to between 15 and 20 times the water concentration within one day of exposure to 1.0 mg/l when equilibrium is reached and the tissue residue concentration reaches a plateau (Tooby, *et al*, 1974; Verloop, *et al*, 1974). The plateau concentration for rainbow trout and roach is not significantly different from that causing deaths after chronic exposure (Tooby, *et al*, 1974). When fish are placed into clean water the dichlobenil residues are eliminated rapidly (Tooby, *et al*, 1974; Verloop, *et al* 1974).

The persistence of dichlobenil in water and mud in field studies has been extensively studied (Van Valin, 1966; Cope *et al*, 1969; Niehuss and Börner, 1971b; Walsh *et al*, 1971; Ogg, 1972; Spencer-Jones, 1974). However, the granular formulation now in use is more persistent than the wettable powders used in the earlier studies. In a field study carried out by the Salmon and Freshwater Fisheries Laboratory a 22% granular formulation of dichlobenil was applied to a pond at a concentration of 1.0 mg/l. Residues in the water increased to about 0.5 mg/l between 1 and 2 weeks after treatment and thereafter decreased to about 0.3 mg/l which was then maintained until 6 weeks after treatment when there followed a further slow decline. Spencer-Jones (1974) found 0.02 mg/l 90 days after an application of a 22% granular formulation of dichlobenil to 1.0 mg/l. Dichlobenil residues at levels above 0.1 mg/kg (wet weight) were found in mud throughout a two-month field study.

Tissue residue levels of dichlobenil follow the rise and decline of residues in water and are accumulated to between 15 and 20 times those levels, as in the laboratory studies. In the Salmon and Freshwater Fisheries Laboratory field studies, roach exposed to about 0.3 mg/l dichlobenil in water accumulated

a plateau level which was not significantly different from the levels found to be lethal in chronic laboratory studies. After five weeks all the fish had died; the gravid females and small males died first. The dissolved oxygen concentration during this period did not fall below about 4 mg/l which was above the lethal level but the respiration rate was probably increased and, therefore, the uptake rate would be greater and a lethal tissue level accumulated more rapidly: Cope *et al*, (1969) reported that growth of bluegills increased after the treatment of a hatchery pond with dichlobenil at 10, 20 and 40 mg/l, using a 50% wettable powder. However, in the pond receiving the highest treatment concentration, 96% of the bluegills died, compared with 40% in the control pond. No stocking density data are given but it is well known that over-stocked populations do not grow as well as under-stocked populations. The surviving 4% probably grew better because of less competition; furthermore, these survivors may have been more resistant fish. There were smaller numbers of fry surviving in the treated ponds than in the control pond. Pathological symptoms developed in the gills of bluegills 4 days after the pond treatment with 0.6 mg/l dichlobenil and the condition of the gills deteriorated further after 63 days (Cope, 1965).

CYANATRYN

This triazine is still undergoing field trials and laboratory studies to determine its suitability as an aquatic herbicide. Cyanatryn has a half-life of about three weeks in water (Roberts, 1974) but residues, in order to kill the target plants, must be maintained at the application concentration for two weeks (Payne, 1974) and is therefore formulated in a slow-release granule to increase the persistence. Suitable micro-organisms can degrade cyanatryn to harmless metabolites.

2,4-D

Uptake and persistence of a C^{14}-labelled dimethylamine salt of 2,4-D in three species of American fish was studied by Schultz (1973). Radioactive residues were found in all fish tissues and organs analysed; however, the actual 2,4-D residue (determined by gas-liquid chromatography) in the muscle tissue was negligible, indicating that most of the C^{14}-residues were one or more metabolites. The major metabolite was thought to be a 2,4-D glucuronic acid conjugate and a high concentration of C^{14}-residue was found in the bile. After an application concentration of 0.8 mg/l to a pond, residues of 2,4-D increased to a maximum concentration of 0.3 mg/l 3 days after treatment and decreased to 0.005 mg/l 2 weeks after treatment (Shultz and Harman, 1974). Very few fish taken from the pond had accumulated detectable residues. No residues were found in mud 56 days after treatment. Robson, (1968) estimated that 2,4-D may take months to degrade in soil while a suitable microflora develops. Few fish deaths have been reported from the use of 2,4-D but Cope *et al*, (1970) found pathological disorders in bluegills after exposure to an ester formulation of 2,4-D and spawning was delayed for two weeks after exposure to 5 and 10 mg/l.

DIQUAT

Grzenda *et al* (1966) found that diquat concentrations in natural water dropped from 2.5 mg/l to 0.01 mg/l after 9 days and was readily adsorbed on to mud. This cationic herbicide can persist in some soils indefinitely but in a biologically inactive form. Diquat did not harm bluegill adults and fry in moderately hard water over a range of 1-3 mg/l, and examination of the fish did not reveal any harmful effects of the chemical and residues in the tissues were absent 12 weeks after treatment (Gilderhus, 1967). However, this herbicide showed a high acute toxicity to cladocerans at the application concentration.

GLYPHOSATE

The maximum concentration given in Table 1 is unlikely to be reached as glyphosate will not be applied directly to water; therefore, there is a wide margin of safety. Furthermore, glyphosate is applied during the autumn. It is totally biodegraded by micro-organisms, and in water bodies the residues are bound to mud particles. In those systems with the lowest potential for micro-organism degradation the half-life of glyphosate, was found to be less than 2 months (Monsanto Ltd., personal communication).

PARAQUAT

No residues could be detected two weeks after application of paraquat (as the Esgram formulation) to a reservoir at a rate of 0.6 mg/l (Brooker and Edwards, 1973). Paraquat is rapidly adsorbed on to mud and, like diquat, very persistent in this bound form. No fish kills were observed in this study (Brooker and Edwards, 1974), but Way *et al* (1971) reported deaths of roach from a reservoir treated with 0.5 mg/l of paraquat; however, these deaths probably resulted from the severe dissolved oxygen depletion and not the direct toxic effect of paraquat.

TERBUTRYNE

Terbutryne appears to be persistent at low concentrations in water for some considerable time. Residues in a pond dropped from 0.25 mg/l to 0.025 mg/l 14 days after treatment and 0.01 mg/l was present 65 days after treatment. A concentration of 0.03 mg/kg was found in the mud 98 days after treatment (Maier-Bode, 1972). However, Van der Weij *et al*, (1971) found that terbutryne residues had a half-life of 25 days in water but 0.02 mg/l was found 161 days after treatment with 0.1 mg/l. They also found 0.18 mg/kg in the organic layers of mud 133 days after treatment. Carp accumulated 0.15 mg/kg in their tissues 7 days after treatment of a pond at a rate of 0.05 mg/l of terbutryne, and thereafter the residues were slowly eliminated following the decline in water concentration; a tissue concentration of 0.01 mg/kg was present after 56 days (Maier-Bode, 1972).

In field studies carried out by the Salmon and Freshwater Fisheries Laboratory, in which a series of ponds were treated with terbutryne at an application concentration of 0.05 mg/l, the dissolved oxygen concentration fell rapidly and remained at a low level for a period of up to four weeks. These low levels were not in themselves lethal to coarse fish, but may have affected the rate of residue uptake.

CONCLUSIONS

The margin of safety between the concentrations likely to be found in water and the 96-hour LC10 for fish is greater than 10 fold for most herbicides when applied at the recommended rate. The persistence in water and fish tissues of those herbicides used against emergent weeds does not appear to present a severe problem for fish survival, but the herbicides used to control submerged weeds and algae are more persistent and may have a long-term effect. Furthermore, some of the herbicides used to control submerged weeds and algae such as dichlobenil and copper sulphate, have a small margin of safety which may be less than 10 fold under certain circumstances. Dichlobenil is accumulated rapidly in fish tissues and, if persistent for sufficiently long enough in the fish, can cause death. Accumulation may be accelerated by increased uptake due to the increased respiration rate which follows the fall in dissolved oxygen concentration in water after weed die-back. This effect may occur with other herbicides in similar depleted oxygen situations and work at the Salmon and Freshwater Fisheries Laboratory is being carried out on those herbicides most likely to cause an oxygen depletion such as the triazines. The triazines (cyanatryn and terbutryne) appear to be persistent in water and fish tissues; furthermore, because they are total herbicides oxygen depletion in the water can be very severe and persist long after treatment. It is quite clear that acute toxicity tests alone do not provide the data required to predict ecological effects and therefore, long-term field studies must be carried out. When attempting to predict ecological effects from laboratory data, discrepancies are likely to occur because of the fluctuating environmental factors affecting the action of the herbicide in field conditions. It is also difficult to assess the risk of invertebrates from the use of herbicides without an extensive long-term ecological study and very few of these have been carried out. Similarly, it is difficult to assess the effects on fish following a change in invertebrate population structure. However, unless all fish-food species are killed, the effect on the fish is likely to be small.

Although relatively high concentrations of these herbicides over a short period of time may not be harmful to fish, lower concentrations over long periods may be harmful and in some cases lethal to fish under some naturally occurring conditions. Provided the herbicide residue uptake does not impair the biochemical and physiological mechanisms by which homeostasis is maintained then there will be little risk to fish. However, there is no easy method available at the present time for measuring significant sub-lethal effects. The toxicity and residue data presented shows that except for copper sulphate and dichlobenil, there will be little risk of acute mortality if the herbicides are used with care, but sub-lethal effects may occur at concentrations 10 fold

or more lower than those used in the toxicity tests or found in the fish tissues. Such low concentrations may not affect fish during the laboratory exposure period or during the field trials but may have a long-term effect; for example, reproduction could be affected which will lead to a depleted population. Experimental procedures are being evaluated in an attempt to assess the effect of low concentration exposure over a long period on reproduction, hatching success and fry survival. The evaluation of such sub-lethal effects from field data is made more difficult because in some cases the impact of weed removal on the fish population is likely to be more severe than the accumulation of residues.

A code of practice has been drawn up by the Ministry of Agriculture, Fisheries and Food, advising users that only a part of any water body should be treated in any one season, allowing areas free of herbicide residues. However, with the new generation of persistent herbicides and the increased scale of use there may be difficulty in keeping some areas free of herbicide residues. Furthermore, those herbicides with a small margin of safety at normal application rates should be used with caution, in order to prevent the risk of damage to fish stocks. The problems arising from the use of aquatic herbicides are complex therefore close collaboration between the Regional Water Authorities, chemical manufacturers and the Ministry of Agriculture, Fisheries and Food is essential.

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